

JOURNAL

OF THE

ALLENTOWN HOSPITAL

ALLENTOWN, PENNSYLVANIA

VOL. 1.

NOVEMBER, 1951

NUMBER 5

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JOURNAL
OF THE
ALLENTOWN HOSPITAL

Published Quarterly

BY THE STAFF AND TRUSTEES
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ALLENTOWN, PENNSYLVANIA

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CHONDROMALACIA OF THE PATELLA

RICHARD K. WHITE, M.D.

Chondromalacia of the patella is a well-defined clinical entity characterized by degeneration of the articular surface of the patella in varying degrees of severity.

ETIOLOGY

The cause of the degeneration may be divided into acute and chronic trauma.

(1) **Acute Trauma:** Direct blows to the patella result in compression of the opposing cartilaginous surfaces of the femoral condyle and the patella. This causes a bruising of the articular cartilage with resultant impairment in nutrition and consequent necrosis.

(2) **Chronic Trauma:** Thinning of the articular cartilage is commensurate with the normal process of wear and tear which occurs with progressive ageing. This has been demonstrated on many cadavers and post mortem specimens. When the articular cartilage is thinned down to subchondral bone, persistence of mechanical friction results in eburnation and proliferative bone reaction. This represents the degenerative or osteoarthritic type of involvement well recognized in the older age group. The initial stages of cartilage degeneration represent the chondromalacic phase of the later-developed degenerative knee joint. Disparity of joint articulation, such as occurs in recurrent dislocating patellas, etc., produces an incongruous articulating surface resulting in excessive wear and tear on the articular cartilage.

PATHOGENESIS

The mechanism of degeneration is directly related to the elasticity of the hyaline cartilage of the patella. The elasticity, in turn, is directly and proportionately related to the chondroitin-sulphuric acid content of the cartilage. With a loss in elasticity, there is a consistent loss in the ability of the cartilage to withstand loading effects. It has been shown that the elasticity of cartilage in malacic or degenerative cartilage is reduced. Hence, it appears that there is a direct relationship between the altered metabolic physiological changes and the production of the pathological condition.

PATHOLOGY

The changes seen in malacic cartilage vary from minimal to severe degeneration, dependent upon the causative factors. Perhaps the earliest change is softening of the articular cartilage. As softening progresses, cracks and fissures appear in the substance of the cartilage. These fissures then widen and tend to become separated from the underlying subchondral bony attachment. As the amount of separation increases, the cartilage becomes further devitalized, and finally separates as small flakes and pieces of dead cartilage. These then tend to lie free in the joint cavity and represent one of the mechanisms by which free loose bodies in the joint may be formed. When a sufficient amount of the cartilage has been removed, subchondral bone then appears. During all these progressive stages, there is an increase in the amount of friction on movement. This is directly related to the reduction in elasticity, as noted above. When the subchondral bone of the patella moves against the articular cartilage of the femoral condyles, new bone is produced by irritation. Osteophytes are thus formed about the area of subchondral erosion. Microscopically, there is an intracellular degeneration of the fibrils, but no specific pathological picture is identified. With the degeneration of a cartilage, there is a concomitant increase in the water content of the cartilage. Normally, the water content is approximately fifty per cent. The increase in water content probably forms the basis for early crepitation and grating often seen in the knee which is otherwise clinically asymptomatic. A pannus tends to form over the remaining portion of the patella cartilage, as well as over the femoral condyles which form the articulating portion with the patella. The synovia shows parallel changes with a severity of the malacic changes. These range from moderate congestion and hyperemia to thickening, hypertrophy, and villous proliferations.

CLINICAL DATA

The patient usually gives a history of either a direct blow on the knee, such as is seen in dash board injuries to the knee cap in an automobile accident, or some other related type of injury. In other cases, the patient gives a history of progressive increase in grating and crepitation. Gradually, the other signs and symptoms develop. In these cases, the degeneration of the cartilage is probably on an intrinsic, chronic, traumatic basis. The symptom complex and pain pattern of these cases usually follows a definite picture. In general, there are the symptoms of internal derangement of the knee, consisting of chronic weakness, tendency for the knee to buckle, joint effusion relative

quadriceps insufficiency, occasional locking and pain. Of more specific interest is the aggravation of pain in inclement weather and the increase in severity of symptoms on coming down the steps. When this latter fact is elicited in the history, it is almost pathpneumonic of disability arising from the patello-femoral compartment. In general, the severity of the symptoms is related to the severity of the condition.

Physical examination shows pain and tenderness related to the patello-femoral compartment. Varying amounts of crepitation, crunching, grating, and creaking, are elicited. Quadriceps atrophy may be present. Compression of the patella against the femoral condyles as the patient moves the knee aggravates the pain. There is usually referred pain over the medial and lateral expansions of the joint capsule. Joint reaction, as manifested by marked effusion, is usually not present. If osteophytes or proliferative bone is present, then clicking or locking may be found. When the patient crosses legs on the affected side, pain may be produced.

X-RAY FINDINGS

As a rule, there is a paucity of positive X-ray material on these cases. Tangential views of the patella may show osteophytes along the lateral margin of the patella. The degeneration and bone erosion is usually not seen. Arthrography with air adds little to the diagnostic problem. Arthrography with opaque material, such as pantopaque, under tangential technique, may show, by contrast, the fibrillation and shredding of the articular cartilage. In general, however, this method does not seem to warrant routine use, since the clinical history and examination are in themselves sufficiently characteristic.

DIAGNOSIS

Given a history of a direct blow to the patella with increasing signs and symptoms, a negative X-ray and positive physical findings, the diagnosis is usually established.

TREATMENT

The treatment, in general, depends upon the severity of the symptoms. Many mild cases can be adequately relieved by immobilization in a walking, cylinder type of cast for four weeks, injection of two per cent novacaine into the joint, and/or the use of intravenous novocaine. When the symptoms are severe, and conservative treatment has proven to be of no avail, surgical treatment is the only method by which relief of pain can be obtained.

There are two methods in use at the present time for this condition. The first is chondrectomy. This procedure consists of shaving the involved portion of the cartilage away until normal cartilage is obtained. The second method consists of extirpation of the patella. It seems logical that if the condition is so severe that operative treatment is necessary, shaving the articular cartilage produces only a temporary improvement in the condition. It is presumed that the changes in the cartilage are already sufficiently far enough advanced to warrant surgical treatment, and, therefore, the method of choice, in my hands, has been patellectomy. If the patella is left in, and only the cartilage shaved, the mechanical factors of stress and strain and continued motion of the patella against the femoral condyles is still present, then the condition will reestablish itself. The good reports following chondrectomy, which have appeared in the literature, do not have a sufficiently long follow-up period to establish this method as a procedure of choice. It would seem inevitable that in a three to five year period, following chondrectomy, the original symptoms would then re-establish themselves.

OPERATIVE TECHNIQUE

A horizontal incision is made over the mid point of the patella, extending approximately one inch on either side. The incision is deepened through the patella bursa down to the substance of the bone. The attachment of the quadriceps tendon into the substance of the bone itself necessitates sharp dissection by means of a knife blade, in order to maintain as much as the quadriceps tendon as is possible. This is carried out by making a vertical incision from the point one inch above the upper pole of the patella to one inch below the lower pole of the patella. The incision thus divides the quadriceps tendon above, and the patella tendon below. Removal of the patella is then carried out by sharp dissection. Imbrication of the vertical incision is then carried out in order to obviate the defect following removal of the patella. A rather tight closure is made in this way, in order that the last five degrees of full extension can be carried out. Otherwise, relative lengthening of the quadriceps tendon occurs, with resultant weakness of the quadriceps mechanism. The skin is closed in routine fashion. A plaster-of-paris cast is worn for two and one-half weeks. Following removal of the plaster, progressive, graded, high-resistive exercises are mandatory, in order to re-establish quadriceps tone and power. In all probability, there is a relative amount of weakness of the quadriceps muscle, principally noted coming down the steps, following removal of the patella. However, this is in direct proportion

to the co-operation of the patient with regards to his rehabilitative training. In addition, this slight defect, should it exist, is negligible in view of the total relief of symptoms which results.

CASE REPORTS

Two cases are selected to demonstrate the pathological and clinical findings. The first case (Fig. 1) demonstrates the marked subchondral bone erosion, fibrillation, and shredded, crab meat-like effect of the articular cartilage. In addition, at the lower pole of the patella, there is a large dessicans, or loose piece of subchondral bone, which was fractured when the patient fell on the ice. Prior to that time he had no symptoms. This patient was an 18 year-old boy who exhibited metabolic changes evidenced by extreme obesity and a yellow staining of his articular cartilage. A tentative diagnosis of ochronosis was entertained, but further studies did not bear this out.

The second case (Fig. 2) represented a 40 year-old woman, who was involved in an automobile accident and struck her knee cap against the dash board. She exhibited many of the signs and symptoms which were enumerated above. The typical appearance of subchondral bone erosion occurring in the mid line, together with the fibrillation and shredding of the articular cartilage is again seen. Both of these cases obtained complete relief of pain and a well-functioning quadriceps mechanism.

SUMMARY

1. Degeneration of the articular surface of the patella is a cause of internal derangement of the knee joint.
2. The pathological picture is one of progressive changes in the articular surface, consisting of softening, cracking, fissure formation, fibrillation, and eventual separation of portions of cartilage.
3. A careful history will elicit characteristic symptoms.
4. Physical examination will demonstrate specific signs.
5. In liability cases, due consideration must be given to the late developments of disability.
6. When the symptoms and signs warrant surgical intervention, patellectomy is the treatment of choice.
7. Post-operative rehabilitation is an important and integral part of the operative procedure.

D-TUBOCURARINE IN WAX AND OIL FOR RELIEF OF MUSCLE SPASM IN TETANUS

HILDA G. RUCH, M.D. and RUTH N. BROWN, M.D.

INTRODUCTION

D-TUBOCURARINE acts by preventing the normal reaction of skeletal muscle to acetylcholine produced at the myoneural junction. Schlesinger (1-2) believes that d-tubocurarine can block the abnormal impulses which cause muscle spasm without blocking the normal impulses which produce muscular contraction, providing the drug can be kept at the proper concentration at the myoneural junction. On the basis of this theory, d-tubocurarine in wax and oil was used at the Allentown General Hospital in two cases of tetanus to relieve muscle spasm.

HISTORY

There are reports in the literature, as early as 1860⁽³⁾ of the use of curare in tetanus. At that time, however, there were no purified extracts of curare and the drug was unreliable and unsafe for use in this disease. Since then many others (4-5) have used aqueous solutions of curare in tetanus. Curare in wax and oil was used in the treatment of tetanus by Schlesinger⁽⁶⁾ in 1946, by Weed, Purvis and Warnke⁽⁷⁾ in 1948, and by Ruch⁽⁸⁾ in 1949 with good results.

Curare preparations have been used in various other conditions accompanied by muscle spasm; acute poliomyelitis⁽⁹⁻¹⁰⁻¹¹⁾, spastic paralysis⁽¹²⁾, muscle spasm in low back pain⁽²⁾, spasm following spinal injury⁽¹³⁾, cerebral palsy⁽¹⁴⁾, rheumatoid spondylitis⁽¹⁵⁾, muscle spasm in rheumatic disease⁽¹⁶⁾, and other spastic states⁽¹⁷⁾.

Other muscle relaxants have been used in tetanus. Tolserol (myanesin or mephenesin) has been used by Berger and Bradley⁽¹⁸⁾, by Towers, Edwards and Wood⁽¹⁹⁾, Berger and Schwartz⁽²⁰⁾, Gammon and Churchill⁽²¹⁾, Davidson Ward and Pask⁽²²⁾, Davidson and Adriani⁽²³⁾, and Boles and Smith⁽²⁴⁾. Goodman and Reinhardt⁽²⁵⁾ used dihydrobetaerythroidine and Campos and Brazil⁽²⁶⁾ used bebeerine dimethyl ether.

AQUEOUS CURARE

Adriani, in 1947, used an aqueous preparation of curare in four patients with tetanus. He found the disadvantages of aqueous curare

in tetanus to be: the short duration of action, the necessity for repeated doses for sustained effect, the closeness of the optimum dose to the paralytic dose, the accumulation of secretions in the pharynx and the loss of the power of deglutition, embarrassment of the pulmonary ventilation by intercostal paralysis, and the necessity for the physician to be in constant attendance.

CURARE IN WAX AND OIL

Many of the above disadvantages are obviated by the use of the wax-oil suspension. The action is even and prolonged and the side actions are few or absent. If properly administered, it is slowly absorbed. If the preparation becomes aqueous (see technique) or if the site of injection is rubbed or traumatized, the rate of absorption will be increased. Rapid absorption may cause symptoms of overdose, therefore anyone using this preparation should be thoroughly familiar with its pharmacology and dangers.

PERTINENT PHARMACOLOGY

The value of curare lies in its muscle relaxing properties; its ability to prevent or minimize muscle response to acetylcholine. Acetylcholine produces a potential by actively depolarizing the myoneural junction. Schlesinger⁽¹⁾ states that this depolarization "reaches a critical value and produces the muscle spike by spreading electronically and depolarizing the neighboring area." Curare prevents this depolarization⁽¹⁾. In using curare for muscle spasm, we take advantage of the fact that, with curare, a partial or critical block can be established which will prevent transmission of abnormal impulses while normal impulses will produce muscular contraction. This is known as the "lissive" action of curare.

Side effects of curare parallel the highly characteristic order in which the various muscle groups are paralyzed.

ORDER OF PARALYSIS	SIDE EFFECT
1. Small rapidly moving muscles; 1. extraocular muscles muscles of middle ear	Double vision Ptosis of lids Increased sensitivity to low tones
2. Muscles of neck; muscles of swallowing muscles of phonation	2. Heaviness of head Tightness of throat Difficulty in swallowing Difficulty in speaking

- | | |
|--|--|
| 3. Muscles of face, particularly those involved in smiling | 3. Marked relaxation of face and jaw muscles |
| 4. Hands, arms and legs | 4. Inability to move fingers, hands and legs |
| 5. Back and abdominal muscles | 5 & 6. Depression of respiration |
| 6. Intercostal muscles | |
| 7. Diaphragm | 7. Respiratory arrest |

ELIMINATION

Curare is detoxified in the liver to some extent but most of it is excreted unchanged by the kidney. There is no evidence that, in the presence of kidney or liver disease, there is cumulative depression although theoretically one would expect this.

PRECAUTIONS

No preparation of curare, whether it be aqueous solution or wax and oil suspension, should be given except when facilities are immediately available to combat respiratory arrest without any delay whatsoever. It is recommended that neostigmine methylsulfate be at hand as an antidote. This is an antidote in a limited sense only, since in gross overdose of curare, neostigmine further depresses. Atropine may be used to decrease salivation. **Inflation of the lungs with oxygen**, through an endotracheal tube if necessary, is the best treatment for overdosage.

DOSAGE

The literature on dosage is very confusing since in many instances⁽⁵⁻⁷⁻¹⁰⁾ millegrams has been stated when units was meant. The first standardized preparation was Intocostrin containing 20 units per cc by the head drop over test. Twenty units of Intocostrin is equivalent to 3 mg. of the active principle, d-tubocurarine as the pentahydrate. Most aqueous preparations contain the equivalent of 20 units or 3 mg. per cc. It will be seen, therefore, that one unit is only .15 mg. or that 1 mg. is equivalent to 6.6 units. To make it more confusing d-tubocurarine in peanut oil with myricin (Squibb) contains 27 mg. per cc or about 180 units; d-tubocurarine in wax and oil (Abbott) contains 30 mg. per cc or about 200 units, while Tubodil (Endo Products) contains 25 mg. per cc or 165 units per cc.

Since we have no means of measuring the curare level, the duration of action and dosage must be judged clinically. According to Schles-

inger⁽²³⁾, dosage depends upon the degree of motor acceleration more than on age or body weight. The most effective dose without loss of motor power is the optimum dose.

Weed, Purvis and Warnke⁽⁷⁾ recommend 3.5 to 4 units of curare in wax and oil per kgm. of body weight but not to exceed 2.5 cc (438 units of the preparation they were using).

In overdose or if too rapidly absorbed, curare in wax and oil will cause difficulty in breathing, increased salivation with tracheal obstruction, dysphagia, generalized weakness and tachycardia. The first sign of approaching maximum dose is ptosis of the lids and diplopia.

TECHNIQUE

Curare in wax and oil must be given meticulously. The points to remember are:

1. Have means of resuscitation at hand; Laryngoscope, endotracheal tube, suction, neostigmine, tracheotomy tray, anesthesia machine or resuscitator with 100% oxygen.
2. Begin with a conservative dose and gradually increase.
3. Give single injection deep intramuscularly.
4. Change site with each injection.
5. Do not repeat in less than 24 hours.
6. Do not massage or allow any trauma at the site of injection.
7. Prevent contact with water which would make a more rapidly absorbable solution.
 - a. warm to 140° (dry heat best)
 - b. protect rubber closures from contact with moisture
 - c. dry needle
 - d. dry syringe
 - e. if preparation is separated at room temperature reconstitute or discard.
8. Shake vigorously to mix and obtain homogenous dose.
9. Inject while warm since the preparation solidifies on cooling.
10. Keep refrigerated.
11. Watch patient continually for 6 hours.

This is potentially a dangerous drug, since we are establishing a depot in the body of many times the therapeutic dose of curare.

REPORT OF CASES

K. M., a 14 year old white boy, was admitted to the Isolation Ward of the Allentown Hospital on September 14, 1949. Sixteen days before admission an abrasion of the knee had been sustained from sliding into base while playing baseball. The knee had appeared to be infected for several days prior to admission and had been treated at home with an antiseptic. He had had no immunizations. Twenty-four hours before admission back pain and spasm had developed.

Physical examination revealed a well developed and well nourished boy with nuchal rigidity, spastic abdominal muscles and back muscles, and a purulent wound on the right knee. The temperature was 99.6F. pulse 100, respirations 20. The blood count and urinalysis were normal.

Course: The patient was placed in a quiet room. The wound was debrided and irrigated. Tetanus antitoxin was given intravenously, intramuscularly and locally, a total of 160,000 units during the first twenty-four hours. 100,000 units of penicillin was given every three hours. Phenobarbital, 180 mgm. every four hours was given for sedation. The following day the patient had frequent and generalized spasms, opisthotonos and trismus. 60,000 units of tetanus antitoxin was given intramuscularly. Sedation was changed to intravenous sodium amytal. On September 16, fluids could not be taken by mouth and convulsive seizures had begun. The patient was placed in an oxygen tent, parenteral fluids were started and 100 units of aqueous curare was given in divided doses. On September 17 the patient's condition was critical. Rectal Paraldehyde in oil was tried for sedation but could not be retained. Intravenous sodium amytal, 225 mg. every four hours, was continued. 110 units of aqueous curare was given in divided doses. On September 18 the first intramuscular injection of d-tubocurarine in wax and oil was given, 0.5 cc. (90 units). The patient was comfortable for about fourteen hours. Then extreme restlessness and severe spasms again developed. 80 mg. per kg. of body weight of Avertin was given rectally to control the spasms. On September 19, 0.7 cc. (126 units) of d-tubocurarine in wax and oil was given intramuscularly. That day the patient was able to turn his head from side to side.

On September 20, 0.9 cc. (162 units) of d-tubocurarine in wax and oil was given and the patient was able to take small amounts of water by mouth. On September 21, 1.2 cc. (216 units) of d-tubocurarine in wax and oil was given after which there was enough relaxation of the jaws to start liquid nourishment by mouth. Sedation was changed to

oral chloral hydrate, 1 gm. every four hours. The dose of d-tubocurarine in wax and oil was increased by 0.1 cc. to 0.2 cc. daily until the optimal dose of 1.6 cc. (288 units) was reached. After five injections of d-tubocurarine in wax and oil the patient was able to move comfortably and was relieved of spasms. The optimal daily dose was continued for nine days. The relaxing effect of the d-tubocurarine in wax and oil was noticeable in one hour after being given and lasted 20 to 22 hours.

The patient was discharged on his 26th hospital day having received a total of 580,000 units of tetanus antitoxin, 10,800,000 units of penicillin and 18.9 cc. (3,402.5 units) of d-tubocurarine in wax and oil. There was complete recovery at the time of discharge.

CASE TWO

G. D., a three year old white boy was admitted to the Isolation Ward of the Allentown Hospital, October 16, 1949. Five days before admission a deep laceration of the scalp had been sustained when the child had fallen out of a moving automobile and struck his head on a rock along side of the road. The wound was cleansed and sutured. No tetanus antitoxin was given and the child had not had any previous immunization.

Physical examination revealed a well developed, well nourished critically ill child with marked trismus, opisthotonos, and generalized muscle rigidity. The wound on the left side of the head was infected.

Blood count showed 77% Hgb; 4,460,000 RBC; 22,600 WBC; 91% Polys and 9% Lymphs.

The child was placed in a darkened, quiet room. Sutures were removed from the scalp, and the wound was debrided and irrigated. The child was placed in an oxygen tent.

Tetanus antitoxin was given locally, 20,000 units, intramuscularly 40,000 units, intravenously by slow drip 175,000 units. Penicillin and streptomycin were started. Sodium phenobarbital intravenously and rectal paraldehyde were given for sedation. 0.1 cc. (18 units) of d-tubocurarine in wax and oil was given intramuscularly. Twelve hours after admission severe convulsive seizures and severe laryngospasms developed. 0.2 cc. (36 units) of d-tubocurarine in wax and oil was given fourteen hours after the first injection. Profuse amount of mucus collected in the pharynx and when the patient was aspirated, laryngospasms developed. Artificial respiration and oxygen, under pressure, were used to restore respirations many times.

At 3:00 A.M., October 18, the child was aspirated. Severe laryngospasm developed and the child died. A spinal tap was done immediately after death. The fluid was normal.

SUMMARY

Curare in wax and oil should not be used for complete paralysis but for its "lissive" action. Doses which prevent convulsions and relieve spasm are of value in cutting down the dose of the usual sedatives, which depress respiration and tend toward pulmonary complications.

Two cases of tetanus are presented in which curare in wax and oil was used for the relief of muscle spasm. Techniques and precautions to be taken in the use of this preparation are discussed.

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PSYCHOSIS WITH INFECTIONS OF THE CENTRAL NERVOUS SYSTEM

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PSYCHOTIC manifestations due to infections or intoxications are not uncommon. The brain and central nervous system are particularly susceptible to some of the bacterial agents or viruses. The meningococcus, treponema pallidum and the virus responsible for lymphocytic choriomeningitis; changes in the electrolyte balance due to dehydration or nitrogen retention, effects of drugs or other poisons may also be associated with neurologic and psychiatric manifestations frequently recognized as delirium. When statistics are appraised as to the prevalence of the various forms of mental diseases, little credit is given to the frequency of these conditions. This may well be due to the fact that such psychoses are mostly benign and transitory. Only a small fraction eventually will require admission to a psychiatric hospital. Since such hospitals are the main sources of the statistics, most of these deliria are never called to their attention and thus escape reporting.

The usual manifestations of such toxic or infectious psychoses include alterations in the state of consciousness with varying degrees of confusion and disorientation. There is evidence of increased psychomotor activity which causes a difficult nursing problem. The stream of thought is associated with shortening and fluidity of the attention, flight of ideas or tendency to repetitiousness and perseveration. Disorders of perception are common and usually consist of illusions or hallucinations. These are frequently visual in type but may well be auditory or involve any of the other senses. Delusional trend, if present, may be paranoid but is usually unsystematized and poorly organized.

Notwithstanding our familiarity with these problems they invariably tax the ability of the hospital personnel to deal with them. The services of a psychiatrist are in urgent demand, sometimes with the hope that he will promptly commit such a patient and thus relieve the hospital of the nuisance. The present trend is to treat the patient in whatever hospital he is admitted. This may mean improving and modifying the existing facilities to render adequate treatment feasible.

While most toxic-infectious psychoses are acute some may assume proportions of a major psychosis with residual symptoms. We are

reporting an example of such a case which we were privileged to observe and follow for over three years.

J. W., a 44 year old, white, male was admitted to the Medical Ward of the Allentown Hospital on January 3, 1948. He was confused, disoriented and unable to furnish his own history. According to his son, one week prior to admission he developed a severe attack of "la grippe" which quickly became "influenza." With this he developed mental manifestations. He was talkative, noisy, restless, would not stay in bed, walked around constantly and was highly euphoric. He thought he was at his place of occupation. Other times he talked about being the Secretary of the Alumni Association of his school making various plans for his group. He named the members of this association but was unable to remember any recent events. He tended to be grandiose. The general picture was not unlike that seen in the manic phase of manic depressive psychosis or paresis.

Further history indicated that he had been in fairly good health until about three months prior to admission. He then complained of fatigue and abdominal pain. He was placed on a milk diet and some medication by his family physician. He was employed by a steel manufacturing company as an inspector. Shortly before Christmas of 1947 he was in a large tank while they were taking X-ray pictures of same. The technician was criticized by the management for having taken these exposures while the patient was in the tank.

He had an equivalent of a high school education. Socially he was a friendly individual, was married and the father of three children. Marital adjustment was satisfactory. There was no history of alcoholism or drugs.

On admission his temperature was 99.2, pulse 72, respirations 30. Within the first day the temperature rose to 101 F. and by the fourth day it became subnormal. The temperature then fluctuated between 97 and 98.4 until the 48th day when it reached 99.4 but became normal before discharge, 55 days after admission. His blood pressure on admission was 110/74.

Systemic review revealed no pathologic findings except for a coarse tremor of the tongue. He was first seen by the Neuropsychiatric Department on the 11th day after admission. At that time sensory examination was unsatisfactory because of the patient's inability to cooperate. He did respond to pinprick and there was no evidence of gross sensory involvement. Cranial nerves: There was no papilledema. Pupils were small, round, equal and reacted sluggishly to light. There

were no demonstrable cranial nerve changes. The abdominal reflexes were not obtained on the right. The deep reflexes were equally increased. There was a questionable positive Babinski on the right but no other pyramidal tract signs. He did not cooperate with the rest of the neurologic examination. He was hyperactive, loud, confused, unkempt and untidy but expressed no hallucinatory trends. The impression was that his symptoms were suggestive of an organic psychosis. Conditions such as virus encephalitis, choriomeningitis or space-taking lesion were considered. He was seen by the eye department the following day. Fundi were normal and there was no evidence of increased intracranial pressure.

Laboratory Studies: Urinalysis showed 14 white blood cells and 4 red blood cells, otherwise negative. Subsequent urinalyses were entirely negative.

Blood count Jan. 5, 1948: Hemoglobin 80%; RBC 4,900,000; Leuk. 5,500; Polys. 70%; Lymphs. 30%. Blood count Jan. 15, 1948: Hemoglobin 80%; RBC 4,650,000; Leuk. 9,000; Polys. 78%; Lymphs. 19%; Eos. 3%.

Coagulation time $3\frac{1}{2}$, prothrombin time 19 seconds, icteric index 9, Van Den Berg negative, cephalin flocculation 2 plus, sedimentation rate 14 mm. 60 minutes. Blood Wassermann, Kahn and Mazzini negative.

SPINAL FLUID STUDIES

<i>Hospital Days</i>	<i>Pressure</i>	<i>Color</i>	<i>Cells</i>	<i>Polys</i>	<i>Lymphs</i>	<i>Prot.</i>	<i>Chlor.</i>	<i>Sugar</i>	<i>Curve</i>
4	240 mm. water	Xantho- chr.	216	5%	95%	245	670	40	
	Smear negative. Culture sterile.								
10		Yellow	275	16%	84%	252	700	46	2225333100 (Meningitic)
	Wasserman Negative.								
18		Color- less	206	11%	89%	158	840	40	2345432000 (Meningitic)
38		Color- less	28	0%	100%	88	680		
55		Color- less	5	0%	100%	107	692		544121000 (Paretic)
1 yr. after hospitalization		Clear	8	0%	100%	82	780	68	Not re- ported.
	Wasserman Negative.								

X-ray examination of the lungs was negative.

Course: The patient presented no specific complaints throughout his hospital stay. He remained euphoric but his overactivity gradually

subsided. His orientation improved partially. His memory remained poor. He could identify nurses, physicians or other patients by sight but could not recall their names. He lost capacity to store new events. He had no recollection of the circumstances leading to his admission or for subsequent events. He was able to recall events prior to his illness although not accurately. There was no tendency to fabrications or confabulations. He became neat in his appearance. Frequently he wandered around the ward during the night. He developed a voracious appetite. He spent much of his time playing solitaire, looking at newspapers and magazines although he could not reproduce any of their content.

Upon discharge, neurologically there were no sensory changes. Cranial nerves were essentially negative. His deep reflexes were equally increased. There were no pyramidal tract signs. There was a coarse tremor of the extended fingers. Station, gait and coordination were unimpaired.

He had been receiving penicillin, sulfadiazine, vitamin B complex plus 100 mgm. thiamin chloride b.i.d., nicotinic acid 100 mgm. t.i.d. Chloral hydrate was employed for sedation. Fluids were administered freely by mouth.

He was subsequently followed through the out-patient department. His wife reported that he slept poorly. Some days he was quiet, but other days talkative and happy. He spent his time listening to radio stories. His memory remained poor, but six months later he could be sent on small errands and made correct purchases. He began to initiate some conversation and take an interest in his children. He gained weight. One year later neurologic examination suggested slight blurring of both disc margins. Pupils reacted to light with hippus. There were coarse tremors of the extended fingers.

Two years later the sleep was still restless and his memory remained poor. Emotionally he became irritable and was subject to brief periods of rage. He gained over 25 pounds. His blood pressure at this time was 190/130. The neurologic signs remained essentially the same.

He was last seen on June 21, 1951 or about 3½ years after the onset of his illness. He continued to eat a great deal and had gained over 30 pounds. He helps at home but forgets his chores and does them over again. He is able to go for walks or to the movies with his children. He is fond of attending baseball games. The patient himself was quiet, passive, but answered promptly when addressed. He felt well and happy. His memory remained poor for remote and

recent events. Counting and calculation were impaired and he was poorly informed about current events. Neurologically, the deep reflexes were equally exaggerated, particularly in the lower extremities, but there were no other positive signs.

COMMENT

This patient was admitted following an acute infectious illness associated with acute psychotic manifestations. These subsided but he developed a residual memory impairment. Neurologic signs were meagre and inconclusive. There were evidences of meningeal and encephalitic changes in the spinal fluid studies. He developed personality changes to the extent that he is now totally incapable of performing any useful occupation.

As to etiologic factors the following conditions were given consideration:

1. Bacterial causes: The possibility of acute meningoencephalitis of infectious origin was discarded due to the absence of adequate febrile changes and the low leukocyte count in the blood, the moderate pleocytosis. That there was evidence of meningitic irritation cannot be denied. The sugar levels in the spinal fluid were at the low normal limit and there was existence of meningitic curve in at least two of the spinal fluid specimens. Syphilitic etiology was ruled out by the consistently negative blood and spinal fluid Wassermann. Tuberculous meningitis is a possibility but the clinical course did not substantiate it.

2. Virus infection: Of these, mumps, measles, chicken pox and equine encephalomyelitis were not considered, because the patient did not have clinical evidence of any of these diseases. Epidemic encephalitis was seriously considered. However, the patient did not present any lethargic manifestations which are common in this condition. His spinal fluid findings showed more excessive type of cellular reaction than usually seen in epidemic encephalitis. Lymphocytic choriomeningitis was perhaps our most serious consideration although it was not proven by special virus studies which were not performed in this case.

3. Involvement due to fungus or parasites, such as toxoplasmosis or cysticercosis or torula meningitis, are the more rare causes. So far as we knew patient had not been exposed to any of these agents nor have we performed any specific tests to prove or disprove their existence. We felt that we were dealing with meningoencephalitis of un-

determined origin. Meningitis because of the pleocytosis and increased albumin content; encephalitis because of the existence of the personality and mental changes.

SUMMARY

Clinical course and laboratory studies were presented of a 44 year old man who was admitted with signs of an acute psychosis. This was apparently induced by meningoencephalitic process of undetermined origin. The acute symptoms subsided but he was left with residual quantitative mental defect.

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Case Report

RHINOPHYMA—EXCISION WITH SKIN GRAFT

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A fifty-nine year old white male school teacher was admitted to the Allentown Hospital on July 26, 1950 and stated that a large "tumor" had been present on his nose for approximately six and a half years. It had grown most rapidly during the past two and a half years but caused him no discomfort except on one occasion one month ago when he had pain in the area which passed up into his head. He described one episode of expressing "pus" and some blood from the mass.

His past, family and social histories were negative except that he had suffered the loss of his right arm in an accident some years ago.

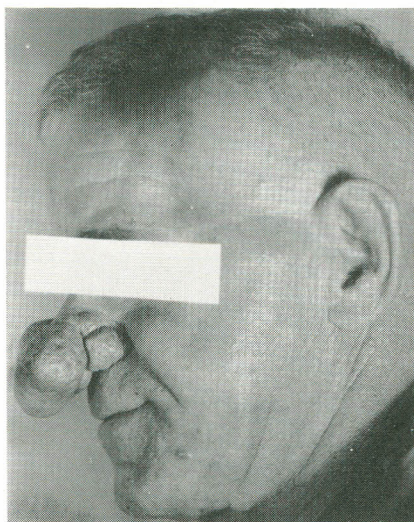
The physical exam revealed a well-developed, cooperative male in good health and excellent general condition except for an old amputation of his right upper arm in its midportion and a bulbous, cauliflower-like swelling on his nose. The growth was firm to the touch, not inflamed or tender, and of a purplish-red color. It was approximately twice the size of a normal nose, and being slightly pendulous, fell downward so that the available nasal breathing space was moderately decreased. The surface of the mass was dimpled and many large pores could be seen on close examination. The picture on the left depicts the patient's appearance at this time.

Preoperative laboratory examination revealed a negative urinalysis a hemoglobin of 86%, a red cell count of 4,940,000, and a white cell count of 6200 with a normal differential. On July 27th, the day after admission, the patient was operated.

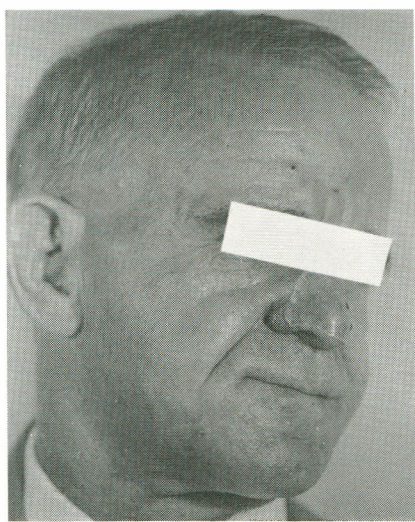
At operation 1% novacaine with ten drops of adrenalin to each ounce was injected into the growth and the mass dissected free from the nasal framework, great care being taken to avoid exposing the nasal cartilages. It was necessary to denude the nose from the distal extremity of the nasal bones to the tip and down each ala to the base. Hemostasis was carefully controlled with ligation and pressure and a thick split-thickness graft was then taken from the medial surface of the left thigh and sutured in place over the raw area of the nose. A stent form of pressure dressing was applied.

Postoperatively the patient received 400,000 units of penicillin daily. His temperature rose to 100 degrees on the fourth day but at all other times was below 98.8. On the sixth post-op day the nose was dressed and a 100% "take" of the graft found. A small pressure dressing was applied and the patient was discharged from the hospital.

The pathological report read as follows: "Specimen comprises a thickened portion of skin approximately 4 cm. in diameter. It has two protruding bulbous masses 4 x 3 cm. each. All skin surfaces are dimpled and the masses are soft. Section shows these to be rubbery, soft, pale pink to grey, edematous connective tissue with several small cystic areas filled with sebaceous material. Microscopic diagnosis: Specimen shows a thin regular squamous surface. The underlying corium contains numerous sebaceous glands. Many of the ducts of these glands are widely dilated to form cyst-like spaces lined with squamous epithelium and filled with keratin. A few clumps of lymphocytes are scattered about. This is a classical picture of a rhinophyma."



Preoperatively



Postoperatively

The picture on the right was taken on September 15, 1950, approximately one and a half months after operation.

The term "rhinophyma" was coined by Hebra in 1856 but the condition was known to the Arabians and had been described by

Hippocrates. However, the cause still remains unknown. Pathologically, modern writers believe the lesion to be a terminal stage of acne rosacea with a profound enlargement of the sebaceous glands and an infiltration of hypertrophic fibrous tissue. The more advanced lesions are best dealt with by surgery with either full thickness or split thickness grafts used as covering.

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